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AN ADDRESS
ON
THE SURGICAL VERSUS THE EXPECTANT
TREATMENT OF INTRACRANIAL
TUMOUR.

DELIVERED AT THE
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BY

SIR VICTOR HORSLEY, F.R.S.,

*Surgeon to the National Hospital for the Paralysed and Epileptic;
Consulting Surgeon, University College Hospital.*

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A SURVEY of the last twenty years' experience of the practical treatment of intracranial tumours shows that the time has arrived for a complete reconsideration of the whole subject.

Probably as a consequence of difficulty in diagnosing (1) the accurate position of the lesion and (2) its precise nature, the whole subject of the treatment of intracranial tumour is unfortunately in much the same false position as that of appendicitis was fifteen years ago.

A few years ago all, and even now most, cases of intracranial tumour died without really receiving any treatment at all, for, since no drug is known to have any influence on neoplastic tissues, the classical administration of potassium iodide, etc., is merely a ritual, and is not treatment of the disease at all.

I am not aware either of any case in which it was proved by *post-mortem* examination afterwards that a cerebral tumour (not one of the specific granulomata) had been cured by drugs during the patient's lifetime. By a singular cynicism, the origin of which I cannot trace further, the unfortunate sufferer from intracranial tumour is commonly said to receive "expectant" treatment.

Considering that in the absence of any active surgical treatment the only thing to be expected is death, the term "expectant treatment" has always to my mind carried with it its own condemnation for inhumanity.

No doubt the expression has arisen from the fact that in so many cases the physician is unable to make a diagnosis on the spot, and while he waits hoping that something will turn up to assist him, he tries to alleviate some of the symptoms of the condition which *de facto* he makes no attempt to treat.

Exactly as in the analogous case of appendicitis, where suppression of symptoms by opiates, etc., formerly caused many a fatality, so in intracranial tumour the temporary betterment which has often followed the empirical administration of potassium iodide and other drugs has constantly led to a fatal postponement of the only real treatment at the present day of a neoplasm—namely, operation. Indeed, this is so constantly done that in the large majority of cases for which operative treatment is sought a careful clinical investigation reveals a clear and unequivocal history of intracranial disease of long duration, from one to even ten years, in one case even more than thirteen years.

The first step, therefore, in reform must be the frank recognition that the so-called treatment of intracranial tumour by drugs is wrong because useless.

The second step must be the enunciation of the earliest pathological conditions which are indicative of intracranial tumour.

At the present time it is usual practice, even when intracranial tumour is suspected, actually to wait until the final stage of the disease is entered upon and the signs of hypertension appear—namely, optic neuritis, headache, and vomiting, and particularly the first of these. My view is that this very common procedure is as unjustifiable as would be the waiting for signs of lymphatic infection in a case of cutaneous epithelioma.

I am of course aware of instances in which the occurrence of optic neuritis was stated to have been the first symptom calling for notice, but I have not myself seen such chronology in a case of intracranial tumour, and would rather suggest that the neurological symptoms had not been observed, an oversight particularly possible in patients of uncomplaining natures.

It is therefore necessary that neurologists should come to some sort of understanding as to the pathological conditions which should be at once treated surgically on the ground that they are indicative of intracranial tumour. These “signal” conditions, to use Seguin’s suggestive expression, may best be discussed according to Dr. Hughlings Jackson’s classification of neurological phenomena—namely, (*a*) those of over-action and (*b*) those of want of action, that is, in respect of the functional activity of the central nervous system.

(*a*) *Over-action*, that is, irritative effects of lesion.

On the first of these, then, I propose that the following procedure should be adopted, namely, “that every case of focalized epilepsy not definitely proved to be idiopathic in origin must be treated by exploratory operation.”

By focalized epilepsy I mean all varieties of epilepsy in which the focus or starting point of the seizure can be localized to one lobe of the cerebrum.

I have elsewhere¹ shown that a diagnosis of the seat of neoplasms can be made by following Dr. Hughlings Jackson’s methods and analysing the mode of epilepsy generated in any one particular region of the cortex cerebri. In order not to take up the time of the meeting and so prejudice the discussion of the main question, the subject of this communication, I must for the present refer my readers to the publication above mentioned.

The character of the epilepsy alone is sufficient as the basis of an exploratory operation, but of course in the large majority of cases the diagnostic conclusion is confirmed by the associated symptoms.

A simple exploratory operation should involve no risks to a patient, and, inasmuch as the successful treatment of a malignant growth depends upon its earliest possible removal, no opposition on a rational basis can be raised against my proposition except this, that in the present state of our knowledge such general resort to operation may lead to negative findings in some instances.

But in such a case no harm would be done to the patient nor would surgical intervention be prejudiced, since the surgeon would have done the best thing in his power for the patient.

In all my cases of such exploratory operations I can only remember one in which the skull and cortex cerebri appeared to be normal when exposed. If the suggested procedure is carried out as a routine practice, it will result not only in the discovery of neoplasms in a very early stage of growth, but will also greatly improve our knowledge of the affections of the brain cortex, which at the present time is chiefly derived from *post-mortem* anatomical examinations, in which only end conditions are found and bearing very little resemblance to the conditions existing during life and of which treatment is attempted.

(b) *Want of action*, that is, destructive effects of lesion.

The second proposition I wish to invite acceptance of is that immediately a destructive lesion of the intracranial nervous system is recognized to have a progressive character it must be treated surgically, and since a rough division of the brain into mainly sensory and mainly motor districts is possible, the following terms express my meaning :

“That every case of *progressive* motor or sensory paralysis of intracranial origin must be treated by exploratory operation.”

The fact that the principal feature of a new growth is its gradual extension and consequently gradual destroying effect on the nerve tissues, does not seem to have received full attention from clinicians. Many, in fact most, cases of intracranial tumour, when referred for surgical treatment, exhibit severe symptoms of destruction, such as hemiplegia, hemianaesthesia, hemianopsia, etc. ; and in some cases, especially where the growth slowly increases and varies in its pressure, as all tumours do, the process of destruction has been allowed to go on for years—three to four years not uncommonly—before the removal of the cause is sought.

This procedure of watching and delay is equally unjustifiable as would be the watching of the gradual destruction of a limb by sarcoma, if such were the common practice ; but fortunately, since in the latter case the tumour is exceedingly obvious, the sufferer enjoys a better chance by being treated in good time. The matter becomes even more serious if the correlative question of cerebral compensation is considered. Personally, I do not think that we are justified in speaking of cerebral compensation of function. The more closely cases of cerebral lesion are studied the more clearly does it appear that the destruction of any given part means a corresponding loss of function, and that it is only the widespread degree of representation which provides that with restricted lesions there shall not be a total or complete loss of function.

Nothing, therefore, is of graver concern to the sufferer from intracranial disease than that it should be attacked before it has had time to destroy any portion of the cerebrum.

Clearly the problem before us is to find what are the earliest symptoms of destruction in the various lobes of the cerebrum. The answer to this is obviously the answer to the question, What is the principal function represented in each lobe ?

What, however, is also needed is a more frequent and systematic clinical inspection of cases. Thus, in the early destructive effects on the

mental powers which is so characteristic of tumours of the frontal lobes, beyond a recognition of the well-known *Witzelsucht*, there exist no organized methods of mental and intelligence examination and record.

So, too, in lesions of the sensori-motor Rolandic region, whether pre-central or post-central, so much attention is bestowed on coarse motor loss that the far earlier loss of Munk's "memories of movements" has been overlooked. I have found that atopognosis may be present for months before the destructive lesion producing it has caused any detectable loss of motor power; whereas stereognosis, on which much reliance is often placed, naturally does not develop until the essential factor in its production—namely, motor paralysis—is well marked.

Thus, as might have been anticipated, the earliest graded progressive signs of destruction to be looked for are losses of sensory function. This is true not only of the central region of the cortex, but *a fortiori* of the so-called higher sensory centres. Thus, early recognition of commencing restriction of the fields of vision, of alteration in tone appreciation, and of slight disorders of equilibration, will secure early treatment of tumours of the occipital and temporal regions.

Though for the early and successful treatment of cerebral tumours operation should be at once resorted to on detection of progressive sensory loss, unfortunately the last phenomenon to appear—namely, loss of motor power—is the one usually looked for, and, one must add, waited for, before the proper surgical steps are undertaken.

It follows from what has been said above that no tumour of the brain should be "treated" by internal drug administration. We come, therefore, to the following conclusion: "Every case of intracranial tumour definitely diagnosed must be treated, according to its situation, either by (1) removal of the neoplastic tissue, or (2) release of the intracranial hypertension."

It might be imagined that the decision as to which of these two procedures should be undertaken is simple, but in the present condition of our diagnostic powers the decision not infrequently has to be left to the moment of operation. Under these circumstances it will be found that the two-stage method of operating I suggested in 1893 gives the necessary freedom of action to the operator and protects the patient from unnecessary risk, though with many of my colleagues I quite agree there is but little risk of shock in removing at one sitting small growths from, for example, the upper parietal region.

The removal of an intracranial neoplasm is, of course, the only means of carrying out the primal duty of the medical adviser of endeavouring to save life, but owing to the special functions possessed by the central nervous system the question to a patient whether he would choose to survive the loss of these is always open.

Indeed, it was owing to a patient deciding before an operation that no procedure involving an increase of his hemiparesis should be carried out, that I made the first observation that a simple decompression operation could cause arrest and complete degeneration of a glioma. Though the same result has been observed since in other cases, they are so few that

it is impossible to draw any conclusions as to the factors which combine to render such a fortunate result possible.

The logical conclusion, therefore, must be that except in the case where the field of removal involves direct destruction of the representation of such a single faculty as speech, free extirpation of neoplastic tissue should be the rule and mere decompression the exception.

Stress may justly be laid upon this point in the present state of this subject, since, in view of the ease with which blindness, etc., can be averted by a simple decompression operation, there is some risk that only palliative operations may be undertaken when curative measures should be attempted.

The question of diminishing the increased tension which the presence of a growing tumour gives rise to within the skull is important, not only because of its immediate results in arresting papilloedema, headache, and vomiting, but also because, as Oppenheim has pointed out, like all therapeutical means, there are in its employment disadvantages to be avoided as well as advantages to be secured, and that both must be considered in discussing means of palliation during the so-called "expectant" treatment.

If the general plan of operative treatment thus indicated is followed, then the first consideration which should be given prominence—namely, the desirability of making the opening in the skull as near as possible to the lesion—is fulfilled. If subsequently it be decided to attempt the removal of the neoplasm, no difficulty will arise in dealing with the surrounding cerebral tissue. The opposite practice not only creates this difficulty but also causes confusion in subsequent attempts to perfect the localization diagnosis.

As regards the practical question of the accidental production of aphasia by reason of brain oedema, that by no means depends upon the situation of the opening, but upon that of the tumour and of its pressure directly interfering with the function of the speech cortex. I am aware, indeed, of an illustrative case in which although a tumour was suspected to lie in the left frontal lobe a decompression was performed on the right side, with the result that aphasia was not only not avoided but followed the operation, no doubt from the direct effect of the growth.

The final point for discussion is the question, Under what circumstances should simple decompression operations be performed?

The importance of deciding this question lies in the fact that such, as already indicated, merely palliative measures may be undertaken, when, on the contrary, curative operations should be entered upon.

Practically the answer can be readily supplied, since there are but two conditions under which a palliative operation is all that should be employed. These are: (1) Cases in which the neoplasm is known to be situated in a position from which it cannot be safely dislodged; (2) cases in which no localization is possible. In both of these classes of cases the execution of a subtemporal decompression is naturally the best procedure.

*Concluding Note on the Usual Procedure in Neurological Practice in
Regard to the Treatment of Intracranial Tumour and Syphilis.*

The whole subject of the "expectant" treatment of intracranial tumour is inseparably connected with that of the treatment of syphilis of the central nervous system, for there can be no doubt that to the empirical treatment of these cases with potassium iodide and mercury is primarily due the desire, in the absence of a correct diagnosis, to try experimentally a course of antisyphilitic treatment. Moreover (from one point of view regrettably), since this treatment is occasionally followed by a modicum of success where diagnosis has failed and the case is really one of syphilitic cerebritis or pachymeningitis, the unfortunate custom has grown up in practice of devoting a long period of months or even years of incalculably valuable time to antisyphilitic treatment.

There are really two points of great importance involved in this question: first the unjustifiability of treatment of non-syphilitic cases with antisyphilitic treatment; and secondly, the justifiability of treating syphilis of the brain by the conventional methods, namely, antisyphilitic drugs given by the mouth, intramuscular injection, or inunction. I wish to take these two points separately, as, though these themes may be thought to be very old ones, there is certainly on the first no definite procedure yet adopted except by a few, and on the second I desire to urge a modification of the present practice in the treatment of syphilitic lesions of the central nervous system.

1. Should any (routine) antisyphilitic treatment be given in cases of intracranial neoplasm, and, if so, for how long?

Unfortunately at the present time, owing to our deficient powers of diagnosing the nature of a lesion, a case presenting the symptoms of an intracranial neoplasm, and yet giving a negative Wassermann reaction, cannot be pronounced definitely and at once to be non-syphilitic in origin.

Consequently, although the case be one of glioma or glio-sarcoma, and therefore in itself uninfluenced (though surrounding oedema may be diminished), probably most practitioners would follow the routine practice of giving iodide and mercury in the hope of producing some effect.

In 1893 I drew attention to this matter, and urged, even in those pre-Wassermann days, that six weeks of such probationary treatment was the utmost that should be allowed; that unless within that period not merely temporary alleviation, but very substantial improvement, had been attained, the case should at once be treated surgically. (I shall discuss directly the value of "improvement" in cases of syphilitic lesion.)

Although the same point was subsequently taken up and argued with great force by Dr. Allen Starr, it does not appear to me that during the seventeen years which have elapsed a rational procedure has been arrived at even now, for intracranial tumours of all kinds, from fibromata to

carcinomata, are still treated not merely for weeks, but even months and year, with iodides and mercury.

The present position is, in fact, one of the gravest reproach to medicine, and reform and progress can only be attained when the writers of text-books make, in their chapters on cerebral tumour, treatment bear a definite relation to diagnosis.

I hope that this discussion will accelerate this, and that a consummation so much to be desired in the interests of the sufferers from intracranial tumour will be arrived at before 1913, or not less than twenty years will have passed since the question was first raised.

2. Is the present mode of treatment of intracranial syphilitic disease adequate?

This second question was originally raised by my teacher, Sir William Gowers, who pointed out the fact that cases of syphilitic lesions of the encephalon are not really cured, though often rapidly and very strikingly relieved, by antisymphilitic treatment, and he showed, further, that after a period of respite the symptoms tend to recur. The relative incurability of cerebral syphilis of itself suggests that the time has come to take more active steps in the matter, and especially at this moment, when Ehrlich has demonstrated that though the acute symptoms can be rapidly checked by an arsenical preparation, this apparently is not applicable to the disease when it attacks the central nervous system.

The lesions I wish specially to refer to are gummata, pachymeningitis (including haemorrhagic pachymeningitis), acute and chronic, and so-called syphilitic optic neuritis.

Though in many cases relief is obtained by drug treatment, in not a few there is no improvement or a relapse occurs. In spite of these facts the position was advanced many years ago by v. Bergmann that syphilitic lesions (he specified gummata) ought not to be subjected to surgical treatment. Partly in consequence of this authoritative statement, but partly, doubtless, because at least improvement was so commonly obtained by non-operative procedures, the question of what principles should underlie our treatment has been rather left out of sight.

I wish, therefore, to advance the view that our present lines of treatment should be remodelled in view of the analogy between the subdural and the intraperitoneal spaces. Upon this basis, and the concurrent requirement of getting as large a dose of mercury as possible in contact with the virus, I have obtained in cerebro-spinal syphilis, just as in tuberculous peritonitis, the most rapid improvement by opening the subdural space and irrigating with sublimate solution (1 in 1,000), even in cases where the ordinary routine drug treatment has failed in spite of a full and fair trial. Further, that since a gumma consists of a wholly necrotic focus of infective material, I suggest it is as important that it should be thoroughly eradicated as a similar tuberculous focus is treated.

Here, again, I would ask also for proof, that a gummatous mass in the central nervous system has ever been cured by mercurial treatment,

whether oral, inunction, or intramuscular. In the absence of such evidence, it clearly is only logical that a gumma cerebri should be extirpated and the subdural space irrigated freely with sublimate lotion. By this means we also secure the most direct treatment of the pachymeningitis which always surrounds a gumma cerebri, and also obtain a more immediate effect on the meningeal (pial) vessels, always in a greater or less degree affected with endarteritis.

I have carried out this procedure in all conditions of syphilitic disease of the encephalon—namely, acute meningitis, chronic pachymeningitis, chronic cerebritis, and gumma—with, in all cases but two, striking benefit. The two fatal cases were both haemorrhagic. In one a large cavity in the membranes, found at the operation to be filled with clot and emptied, was never obliterated—that is, the brain failed to recover from prolonged compression (the patient had been hemiplegic for some weeks); and in the other the haemorrhage had already invaded the ventricles.

Thus the procedure involves no risk in itself. Hence this line of treatment, though it may appear at first sight to be more “heroic” than the ordinary methods, as a matter of fact is not so.

Syphilitic Optic Neuritis.—The occurrence of optic neuritis in a syphilitic subject is frequently regarded as a local manifestation of the infection and treated as such. I wish to suggest for discussion whether such optic neuritis is not always a concomitant of subacute meningitis. A near parallel to such a condition is the optic neuritis observed in children suffering from otitis media.

With this view of the pathological causation of syphilitic neuritis, it is easy to understand that opening of the subdural space and irrigation with 1 in 1,000 sublimate lotion offers the best opportunity of a rapid cure of the condition.

This is the case, and a simple subtemporal irrigation on one side will secure immediate, and as far as I have seen permanent, subsidence of the papillitis. Though “syphilitic optic neuritis” often clears up satisfactorily, it has the characteristic tendency of the disease to relapse, and also, owing to its chronic duration under ordinary treatment, leads to a certain degree of atrophic injury of the nerve fibres. The irrigation method of treatment meets both these contingencies in the most efficient manner.

I have previously directed attention to the beneficial results of treating spinal pachymeningitis cases by the irrigation method, and would now conclude by pointing out that in all syphilitic conditions of the nervous system the problem of rapid treatment is the same, namely, the local application of mercury, and that this can be safely accomplished by subdural irrigation.

REFERENCE.

- ¹ *University Hospital Medical School Magazine*, No. 1, 1910.

